Key words: lumbar, retroperitoneal, skeletopy, syntopy.

306. DYSLIPIDEMIA IN PATIENTS WITH TYPE 2 DIABETES ACCORDING TO THE DEGREE AND TYPE OF OBESITY.

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Introduction. By 2025, worldwide, approximately 300 million people is estimated to have type 2 diabetes. In 72-85% of cases the type 2 diabetes is Associated with atherogenic dyslipidemia, the important cardiovascular risk factor. It is characterized by elevated levels of TG, LDL-C and decreased level of HDL-C.

Objective of the study. Comparative analysis of the atherogenic lipid profile in patients with type 2 diabetes based on the degree and type of their obesity.

Material and methods. A retrospective, descriptive and analytical study was realized on 194 patients with type 2 diabetes, hospitalized in Republican Clinical Hospital, Department of Endocrinology, during february 2015-january 2016.

Results. The study included 83 men (42.8%) and 111 women (57.2%) with mean age of 56.2 ± 0.4 years and mean duration of diabetes of 10.5 ± 0.3 years. Patients were divided into 5 groups according to BMI: normal weight-25 patients (12.8%), overweight-52 patients (26.8%), obesity grade 1-63 patients (32.5%), obesity grade 2-39 patients (20.1%), obesity grade 3-15 patients (7.7%). The lipid profile of the groups was: to the group with normal weight: TG = 2.06 ± 0.05 mmol / l, LDL-c = 4.3 ± 0.08 mmol/l, HDL-c = 1.079 ± 0.01 mmol/l. Group with overweight: TG = 3.11 ± 0.16 mmol/l, LDL-C = 5.25 ± 0.14 mmol/l, HDL-C 1.04 ± 0.02 mmol/l. Obesity grade 1: TG = 3.69 ± 0.22 mmol/l, LDL-C = 5.62 ± 0.13 mmol/l, HDL-c = 1.002 ± 0.03 mmol/l. Obesity grade 2: TG = 4.47 ± 0.18 mmol/l, LDL-C 6.51 ± 0.10 mmol/l, HDL-C 0.99 ± 0.02 mmol/l.Obesity grade 3: TG 6.31 ± 0.29 mmol/l, LDL-C = 7.42 ± 0.45 mmol/l, HDL-C 0.76 ± 0.02 mmol/l. So, a statistically significant difference was obtained ($p < 0.001$) between the degree of obesity according to the lipid panel. Also the atherogenic index was calculated, which tended to increase depending on the degree of obesity, the highest value recorded to obesity grade 3 = 14.70 ± 0.91.

From the total amount, 122 patients (62.8%) have android obesity and 72 patients (37.2%) have gynoid obesity. Each group of 5 was divided into 2 subgroups according to type of obesity: android or gynoid. A statistically significant difference was obtained ($p < 0.05$) between subgroups according to lipid profile.

Conclusions. In the lipid profile of patients with type 2 diabetes exist the difference according to BMI, a great severity of dyslipidemia is directly proportional to the degree of obesity. The type of obesity it's very important, more serious disorders of lipoprotein metabolism is registered in patients with android obesity.

Keywords: Type 2 diabetes mellitus, dyslipidemia, obesity.

307. GENETIC ASPECTS OF PRADER-WILLI SYNDROME

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Introduction: PWS is a complex genetic disorder affecting appetite, growth, metabolism, cognitive function and behavior.

Characterized by: 1) Low muscle tone 2) Short stature (when not treated with growth hormone) 3) Incomplete sexual development 4) Cognitive disabilities 5) Behavioral problems 6) The hallmark characteristics – chronic feelings of insatiable hunger and a slowed metabolism that can lead to excessive eating and life-threatening obesity.

The syndrome is due to the loss of expression of several genes encoded on the long arm of chromosome 15 (15q11.2–q13). The complex phenotype is most probably caused by a hypothalamic dysfunction that is responsible for hormonal dysfunctions and for absence of the sense of satiety.

People with PWS have a flaw in the hypothalamus part of their brain, which normally registers feelings of hunger and satiety. While the problem is not yet fully understood, it is apparent that people with this flaw never feel full; they have a continuous urge to eat that they cannot learn to control. To compound this problem, people with PWS need less food than their peers without the syndrome because their bodies have less muscle and tend to burn fewer calories.

Materials and methods: While doing the review we screened worldwide literature and interactive sources. We tried to choose information that will be reliable and will explain the genetic aspects, clinical features and complications of PWS.

Discussion results: PWS is rarely seen worldwide disease especially in RM that imposible diagnoses without genetics tests in neonates. Increasing awareness to PWS can bring to increase use of genetic methods of diagnoses and less miss diagnoses.

Conclusion: Prader-Willi syndrome is a complex multisystem disorder. Patients can be affected by various problems; therefore precocious diagnosis is fundamental to guarantee optimal assistance. Each patient should undergo personally tailored treatment from birth. Therapeutic decisions and clinical followup need to consider all of these possible problems. A multidisciplinary team is required, made up of specialists such as neonatologists, geneticist, pediatricians, endocrinologists, orthopedic surgeons, psychologists, psychiatrists, physiotherapists, and urologists to deal with the numerous medical and psychological problems a PWS patient has to face. Only in this way we can improve quality of life, prevent complications, and prolong life expectancy in patients with PWS.

Key-words: Prader-Willi, Obesity.